has been only one other 80% restenosis in the 70-odd patients who have been treated after that time. We looked into the subset. In the paper we have, indeed, reported log-rank comparisons between survival curves of the Wallstents that we were using originally and the nitinol stents that we are using now, as well as whether there was a difference between post-CEA restenosis patients, whom we were treating initially, and primary high-risk patients with symptoms. There were no significant differences; however, the numbers were too low to make a definitive determination. We suspect it has to do with the type of stent we have been using.

Dr Luis A. Queral (Baltimore, Md). I'm wondering if you can comment on whether it's justified to put patients with recurrent artery stenoses with primary disease in the same group, inasmuch as the pathogenesis seems to be somewhat different. I also wonder whether the type of endarterectomy performed on 65% of these patients influenced the recurrence rate, whether it was patched or not patched, a reversion. And I know that's an old question in vascular surgery, but it has an impact here since that's the majority of your patients. And finally, can you relate the recurrence rate to the location of this restenosis? It seems to me that it might be a different thing to have a recurrent stenosis in the common carotid proximal to the previously treated lesion versus one in the internal carotid artery above the area of surgery.

Dr Lal. I think that's an excellent question. Restenotic lesions within a 36-month period after CEA are usually secondary to intimal hyperplasia. And several authors have speculated that a stent in such a lesion may induce higher rates of in-stent restenosis.

We compared the in-stent restenosis rates of our post-CEA restenosis patients with our primary lesion patients and haven't noticed any difference. It may just mean that the number of patients that we have in our group right now is too low to identify that difference. And that's probably the best that can be said right now in answer to your question. Unfortunately, some of the publications with large numbers of patients have not followed their patients beyond 6 months.

Dr Linda Harris (Buffalo, NY). Have you looked at the subset of patients with the primary atherosclerotic disease, which is a different entity than restenotic lesions? What is your follow-up on those individuals, and on the basis of that, can we say anything about stenosis in those individuals with carotid stenting or not?

Dr Lal. The mean follow-up for our primary patients is approximately 16 months, so it's not very different from the mean follow-up of our entire cohort. And again, the in-stent restenosis rates between our primary and our restenotic patient groups are not significantly different as yet. Two of our patients out of the hemodynamically significant five patients had primary lesions and three had restenotic lesions.

INVITED COMMENTARY

Kenneth Ouriel, MD

The article by Lal and colleagues represents an important contribution to the accumulating body of knowledge upon which clinical decisions concerning carotid angioplasty and stenting will be made. Uniquely, this study defines the long-term incidence of in-stent restenosis and its clinical relevance. The authors concluded that restenosis after carotid stenting is both an infrequent event (6% at 5 years) and one rarely associated with neurologic sequelae (0/5 restenoses). For sure, Lal's study has limitations. The duplex criteria for restenotic lesions appear to be validated for primary, nonstented carotid arteries and not for either recurrent lesions after endarterectomy or for in-stent restenoses. The number of evaluable patients at 5 years is small (four patients at the 60-month time point), the study population is skewed toward patients with restenosis after carotid endarterectomy, and a majority of patients were treated with a stent that many would consider outmoded. While subgroup analyses failed to identify relationships between these variables and outcome, the statistical power of these analyses was low. Importantly, the infrequent occurrence of restensosis (5 of 122 cases) accounts for much uncertainty in the estimate of restenosis-associated neurologic events. The observed frequency of zero events in five restenotic patients does not exclude a true rate of symptoms as high as 45% (95% confidence interval, 0%-45%).

These limitations aside, Lal's study represents yet another piece of the carotid stenting puzzle, providing data on the durability of the procedure. Restenosis after carotid stenting does not appear to occur with the frequency observed in smaller vessels such as the coronary arteries. Rather, the rate of restenosis after carotid stenting appears analogous to that following carotid endarterectomy; restenosis occurs infrequently and when it does, it often develops silently and without symptoms. Taken in concert with the accumulating body of evidence on the short-term safety of stenting, the restenosis data add to the realization that percutaneous carotid interventions are often reasonable alternatives to standard carotid endarterectomy. Although this is a finding that few surgeons would have predicted, failure to embrace this evolving technology risks exclusion from participation in the care of patients with carotid disease. On the other hand, involvement in the ongoing investigations of carotid stenting will, in the words of Winston Churchill, ensure that "history will look favorably upon us, for we will write it."